

## A Case of Late Stent Thrombosis Following Platelet Transfusion in a Patient With Aplastic Anemia

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Aplastic anemia is a condition in which the bone marrow fails to produce adequate numbers of peripheral blood elements. The incidences of atherosclerosis and myocardial infarction in patients with congenital coagulation disorders and chronic thrombocytopenia are very low. In this paper, a case of late stent thrombosis within a drug-eluting stent occurring after platelet transfusion in a patient with aplastic anemia is presented. The authors' observations emphasize the risks of platelet transfusion and the authors' support withholding such a treatment unless vitally indicated, in patients with coronary artery stent implantation and even in those on dual antiplatelet therapy. (**Korean Circ J 2012;42:54-57**)

**KEY WORDS:** Stent; Thrombosis; Platelet transfusion; Aplastic, anemia.

### Introduction

Aplastic anemia is a form of anemia in which the bone marrow fails to produce adequate numbers of peripheral blood elements. The incidences of atherosclerosis and myocardial infarction in patients with congenital coagulation disorders and chronic thrombocytopenia are very low. However in cases requiring invasive procedures (e.g., dental procedure and endoscopic procedure), patients at times need platelet transfusion. In this paper, a case of late stent thrombosis was reported within 4 hours after transfusion of platelets for anticipated bleeding in an aplastic anemia patient.

### Case

A 52-year-old man who had been diagnosed with aplastic anemia

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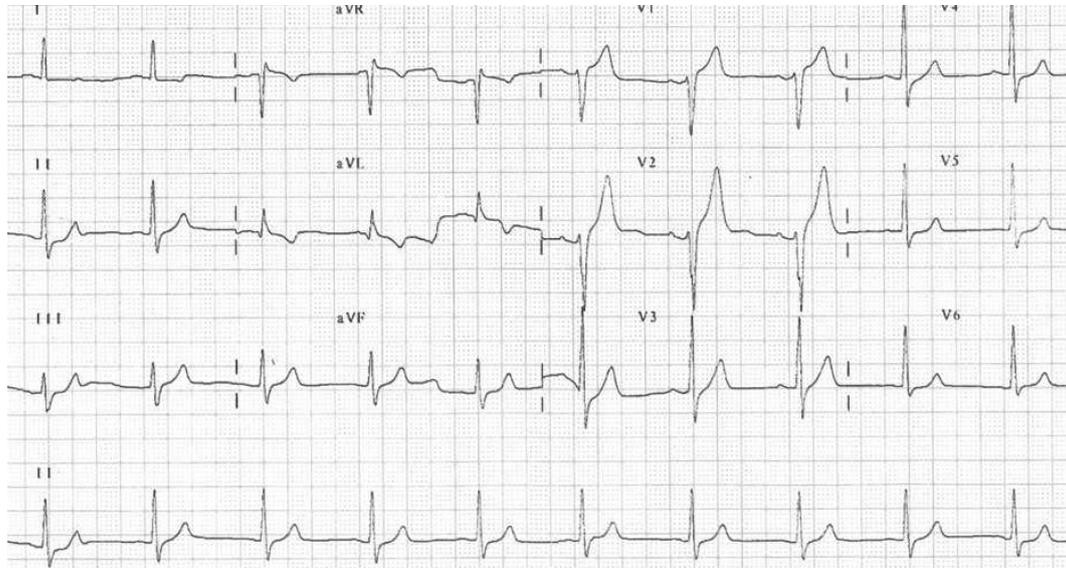
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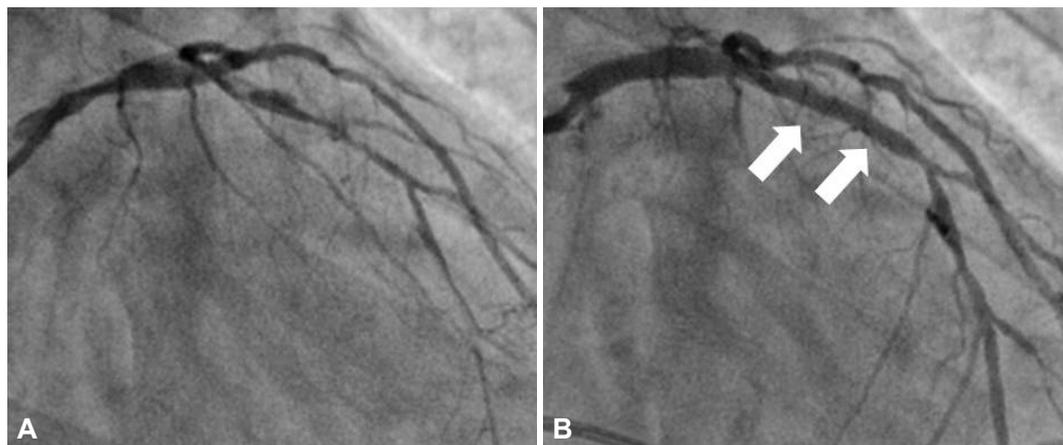
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and stable angina visited the emergency room of the authors' hospital with a complaint of chest pain. The chest pain had developed 1 hour before the patient's arrival at the hospital. The initial electrocardiogram showed a hyperacute tall T wave in the precordial leads (Fig. 1). In the initial laboratory findings, the cardiac markers were in the normal range, but thrombocytopenia was noted (initial white blood cell count: 3350, hemoglobin: 12.5 g/dL, platelet: 45000/ $\mu$ L). The patient's vital signs were stable. His past history revealed that he had been admitted to a hospital a year ago for acute coronary syndrome and percutaneous coronary intervention (PCI) of the left anterior descending artery (LAD), for which a Cypher Select stent (3.5 $\times$ 28 mm, Johnson & Johnson-Cordis, Miami, FL, USA) procedure was performed (Fig. 2). He had been taking 100 mg of aspirin, 75 mg of clopidogrel and 100 mg of oxymetholone (anabolic steroids) for aplastic anemia daily since the last one year. However, he had stopped taking aspirin and clopidogrel for 3 days before presenting himself at the hospital since he was scheduled to be treated for a decayed tooth. He also visited the hematology clinic routinely. His laboratory findings showed that he had severe thrombocytopenia (platelet count: 12000/ $\mu$ L). His hematologist recommended platelet transfusion for anticipated bleeding. Therefore, transfusion of 6 pints of platelet concentrates was performed on the same day of the patient's visit to the hospital. Four hours later, he developed chest pain.

The emergent coronary angiography demonstrated total occlusion of the mid-LAD. Balloon angioplasty was done with a 3.0 $\times$ 15 mm non-complaint coronary balloon. Thrombolysis in myocardial infarction 3 distal flow was obtained. Thus, no additional stent was



**Fig. 1.** Initial electrocardiogram showed hyperacute tall T wave in the precordial leads.



**Fig. 2.** One year before this event, coronary angiography revealed a diffuse, eccentric luminal narrowing up to 90% (A) and percutaneous coronary intervention with Cypher Select 3.5x28 (Johnson & Johnson-Cordis, Miami, FL, USA) was performed due to acute coronary syndrome (arrows) (B).

needed (Fig. 3).

A dose of 7,000 units of unfractionated heparin was injected during the CAG with another loading dose of 300 mg of clopidogrel. After the PCI, the chest pain subsided. On the second day, blood started oozing from the puncture site, hence a manual compression dressing was applied for 1 hour, after which there was no more complications. The follow-up platelet count was 12000/ $\mu$ L. On the 12th day, the patient's dental caries was treated with tooth extraction without bleeding complications, and the patient was discharged. He was prescribed triple antiplatelet therapy with aspirin, clopidogrel, and cilostazol. He was also asked to continue oxymetholone (anabolic steroids) therapy for aplastic anemia.

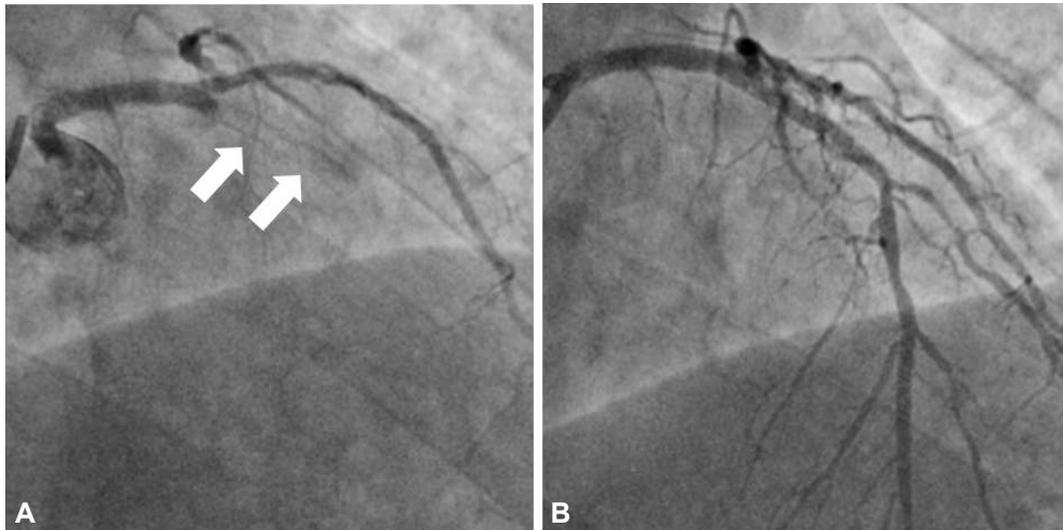
## Discussion

Aplastic anemia is a form of anemia in which the bone marrow

fails to produce adequate numbers of peripheral blood elements. The incidences of atherosclerosis and myocardial infarction in patients with congenital coagulation disorders and chronic thrombocytopenia are very low.

There are a few reports of cases of aplastic anemia associated with myocardial infarction during treatment with anabolic steroids.<sup>1)</sup> Steroids have been commonly used to treat aplastic anemia, are a possible risk factor for coronary thrombosis, because steroids have an accelerating effect on metabolic changes and can induce a hypercoagulable state, each of which may promote atherosclerosis. Therefore, steroids may have helped to precipitate the clinical events in the patient.<sup>2)</sup> Andrade et al.<sup>3)</sup> reported a case of acute stent thrombosis in a patient with giant cell arteritis, but there is no case report of long-term oral steroid therapy associated with very late stent thrombosis.

Even if other risk factors for stent thrombosis (e.g., incomplete



**Fig. 3.** Coronary angiography of the left coronary artery. Total occlusion of the mid left anterior descending artery in the portion of the previously inserted stent (A) was noted (arrows). After balloon angioplasty with 3.0×15 mm non-compliant coronary balloon, thrombolysis in myocardial infarction 3 distal flow was obtained (B).

stent expansion or clopidogrel resistance), according to time sequence, could not be completely excluded, platelet concentrate transfusion might have played an important role in coronary stent thrombosis in this study. Sabovic and Simona<sup>4)</sup> suggested that a substantial number of platelets and whole blood transfusions might be risk factors for coronary thrombosis. That is, plasminogen activator inhibitor-II, a strong prothrombotic factor, which accumulates in stored blood products in a time-dependent manner, could extensively increase in the plasma after transfusions. This prediction, which may be clinically relevant, must be further clarified.<sup>5)6)</sup> Unfortunately, this marker or other coagulation factors have not been examined.

There are few reports of stent thrombosis associated with blood component transfusion. Cornet et al.<sup>7)</sup> reported three case studies of coronary stent occlusion after platelet transfusion. Actually, since peri- or intra-procedural massive bleeding was noted in all three cases, platelet transfusion was done inevitably. In our case, one could argue that platelet transfusion was not necessary in a patient who was scheduled for a minor dental procedure.

The use of intravenous immunoglobulin (IVIG) concomitant with platelet transfusion is indicated in patients with life-threatening severe thrombocytopenia or hemorrhage, or when therapeutic procedures with some risk of bleeding are performed.<sup>8)</sup> IVIG should be administered only after careful consideration of the risks and benefits, and should be reserved for patients at risk of severe hemorrhage, and the use of a bare-metal stent over a drug-eluting stent should always be considered as the first option.<sup>9-11)</sup>

In conclusion, the authors' observations emphasize the risks of platelet transfusion and support withholding such a treatment unless vitally indicated, in patients with coronary artery stent implantation and even in those on dual antiplatelet therapy.

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